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Review

Factors and processes causing accelerated decomposition in human cadavers – An overview

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ABSTRACT

Artefactually enhanced putrefactive and autolytic changes may be misinterpreted as indicating a prolonged postmortem interval and throw doubt on the veracity of witness statements. Review of files from Forensic Science SA and the literature revealed a number of external and internal factors that may be responsible for accelerating these processes. Exogenous factors included exposure to elevated environmental temperatures, both outdoors and indoors, exacerbated by increased humidity or fires. Situations indoor involved exposure to central heating, hot water, saunas and electric blankets. Deaths within motor vehicles were also characterized by enhanced decomposition. Failure to quickly or adequately refrigerate bodies may also lead to early decomposition. Endogenous factors included fever, infections, illicit and prescription drugs, obesity and insulin-dependent diabetes mellitus. When these factors or conditions are identified at autopsy less significance should, therefore, be attached to changes of decomposition as markers of time since death.

1. Introduction

Following death, a complex series of biochemical and pathological processes are initiated that result in considerable alteration of the structure and composition of the human body.[1] Given that many of these changes occur sequentially, it has been proposed that evaluation of the types and degree of changes present may enable estimation of the time since death.[2] Although the most reliable of these postmortem changes has been body core temperature, it is recognized that these changes are quite variable, and that such estimates are approximate at best. Despite considerable individual differences, however, other factors such as livor mortis, rigor mortis and decomposition have been used to give some indication of the time since death.[2] In the following review factors and processes that may cause accelerated decomposition are analyzed, as these may confound estimates of postmortem interval and raise questions as to the veracity of witness statements.

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2. Materials and methods

Case files at Forensic Science SA, Adelaide, South Australia, were retrospectively reviewed for cases where the degree of decomposition noted in the autopsy report appeared greater than would be expected for the time interval since death. Decomposition was assessed by the presence of green/red discoloration of the skin with venous marbling, fluid-filled blister formation, slippage and purging of putrefactive fluids from the nose and mouth. Additional findings included distension of subcutaneous tissues and body cavities by gas from bacterial action, with an offensive odor and putrefactive changes of internal organs with effusion formation. The literature was also reviewed for similar cases.

3. Case reports

Case 1: A thin 14-year-old girl (BMI 11.8) with a history of intellectual disability and insulin-dependent diabetes was found dead in her bed beneath a blanket and a thin quilt at around 1900 h. She had been unwell in the days preceding death. Despite having been alive in the early afternoon the body demonstrated greater putrefactive changes than would be anticipated, with sunken eyes and marked greenish putrefactive discoloration of the abdomen. The ambient temperature was 18 °C and there was no electric blanket or heating in the room. At autopsy the internal organs showed changes of early

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decomposition. Death was due to dehydration and hyperglycemia (vitreous glucose level = 31.1 mmol/L) complicating insulin-dependent diabetes mellitus.

Case 2: A 74-year-old man was found dead at his home address a day after he had spoken to relatives by telephone. At autopsy the body demonstrated greater putrefactive changes than would be anticipated, with venous marbling, green discoloration, purging and putrefaction of internal organs. Death was due to perforation of a small intestinal diverticular abscess with peritonitis.

Case 3: A 56-year-old man was found dead at his home address having been seen alive the day before. There was a history of morbid obesity with a BMI of 77. At autopsy the body demonstrated greater putrefactive changes than would be anticipated with purging of putrefactive fluids from the mouth and nose, putrefaction of internal organs and putrefactive effusion fluid accumulation in the chest and abdomen. A low-grade glioneuronal tumor was found in the right frontal lobe.

None of the above cases involved exposure to excessive environmental temperatures.

Case 4: A 51-year-old man was found dead in a water-filled spa having drowned following an overdose of methylamphetamine. He had been submerged in the warm spa water for a number of hours. The spa water also contained a significant amount of fecal material. At autopsy the body demonstrated far greater putrefactive changes than would be anticipated with green discoloration of the face and body with scrotal swelling. There was extensive skin slippage with purging of putrefactive fluid from the mouth and nose. All internal organs were markedly putrefied.

4. Discussion

The above cases demonstrate enhanced putrefactive changes in cases of diabetes mellitus, morbid obesity and sepsis, in addition to a situation where an individual was exposed to increased environmental temperatures. A range of other specific circumstances where there may be an apparent disparity between the extent of the decompositional changes and the proposed interval since death have also been reported in the literature. In such cases a pathologist may be requested to attend a death scene, given concerns raised by the police of an apparent discrepancy between the alleged time that the victim was last seen alive and the extent of decomposition.

After death, certain changes develop in bodies that include livor mortis, rigor mortis, algor mortis, autolysis and putrefaction, followed by saponification, mummification, and skeletonization.[2] Several of these changes occur concurrently and may provide valuable information to investigators regarding the approximate time of death. Decomposition refers to the process of tissue breakdown and is contributed to by the interaction of autolysis and putrefaction, often with superimposed insect activity.[1] However, a number of different factors may accelerate the rate of decomposition, making estimation of the time that has elapsed since death difficult.

Autolysis begins within minutes of death and represents the process of self-digestion from the activity of endogenous enzymes.[3,4] Cessation of blood flow leads to the accumulation of waste products including lactic acid that damage cells. Plasma and cytoplasmic pH reduce, compromising cell and lysosomal membrane integrity.[2,3,5] The resultant rupture of lysosomal membranes leads to the release of enzymes such as proteases, lipases and amylases into the cell cytoplasm with breakdown of carbohydrates, fats and proteins. Eventually cell membrane destruction releases nutrient-rich fluids that contain amino acids, sugars, glycerol, and fatty acids, all of which serve as food and energy sources for micro-organisms, facilitating putrefaction.[3,5] Autolytic changes progress, therefore, more rapidly in organs and tissues that are lysosome-rich with a high hydrolytic enzyme content, such as the liver, pancreas, spleen, and lungs. Organs with high water content such as the brain are also more susceptible to autolysis.[3,4]

The rate of autolysis and putrefaction may be altered by the ambient temperature (and also by the internal core temperature at the time of death), with autolysis developing faster at higher environmental temperatures and with antemortem fever.[2] This was a factor in a number of deaths that occurred during a heat wave in South Australia in early 2009. Accelerated hydrolysis of tissues causes further protein breakdown leading to the accumulation of basic nitrogen products, such as ammonia and amines. These neutralize the acids resulting from carbohydrate metabolism, and eventually raise tissue pH to alkaline levels, a situation highly favorable to bacterial growth and enhanced putrefaction.[5] This occurs in individuals who have been left outside exposed to the sun or to temperatures above 40 °C, or where victims have died, or become incapacitated in houses during heat wave conditions or where central heating has been left on. Similar problems arise when victims have died in hot bath water (as in case 4), under hot showers, in beds with excessive bedding/electric blankets, or in front of heaters. An additional factor enhancing bacterial growth in case 4 was immersion of the body in warm water containing fecal material.

On occasion there may be differences in the degree of putrefaction in different parts of the body if there has been exposure to different temperatures. An example would be of marked putrefaction of the head compared to the body if the head had been in full sun and the body lying in shade. This 'regional putrefaction' may be useful finding in attempting to reconstruct the position of a body.

Accelerated decomposition is also often a feature in cases where individuals have died inside automobiles, for example from carbon monoxide toxicity.[1,9] In the latter instance elevated cabin temperatures are contributed to by both hot engine exhaust gases and heating of the vehicle by the sun through windows. Bodies in fire deaths may also decompose quickly due to the high ambient temperatures.

The most common putrefactive organisms are anaerobic spore-bearing bacilli, coliforms, diphtheroids, micrococci, and proteus species, that are normally found as commensal bacteria in the respiratory tract and intestines. Putrefactive bacteria predominantly originate from the intestinal tract with *Clostridium perfringens* being the most common and significant species.[6] Autolysis of internal organs such as the intestines facilitates the translocation of bacteria into the blood stream through which spread can occur to tissues all around the body.

Reductive catalysis by endogenous bacteria produces sulfur-containing amino acids (cysteine, cystine, and methionine) which yield hydrogen sulfide (H_2S).[2] Hydrogen sulfide then combines with haemoglobin released from autolytic erythrocytes to produce sulfhaemoglobin, which is responsible for the external greenish discoloration that is usually the first sign of putrefaction. This most often occurs in the abdominal wall of the right iliac fossa overlying the caecum which is fluid-filled and rich in bacteria.[7] This process is enhanced by elevated exogenous and endogenous temperatures and so usually appears between 12 and 18 h in summer and 1–2 days in winter.[8]

Putrefactive bacteria spread most easily in fluid and thus initially spread to, and cause discoloration of, the more moist areas of the body (e.g. dependent areas affected by oedema) and also within the venous system leading to haemolysis and staining of vessel walls and adjacent tissues; i.e so-called 'venous marbling'.[8] Deaths where there are open wounds or injuries to the body, such as burns or lacerations, may also putrefy at an accelerated rate due to breaching of the skin barrier providing a portal of entry for exogenous bacteria.[6]

Algor mortis refers to postmortem cooling and is a useful predictor of postmortem interval. After death heat production stops, but heat loss to the environment continues via conduction, convection, radiation, and evaporation.[4] Since bacteria are using the body as a growth

medium, the most important factor determining the rate of putrefaction is the temperature of the body after death. Thus, the slower the rate of algor mortis, the faster the rate of putrefaction. High initial body temperature at the time of death may be due to physical activity, heat stroke, central fever (e.g. stroke, intracranial haemorrhage), hyperthyroidism, malignant hyperthermia, neuroleptic medication, or intoxication with illicit drugs.[2,8,10–12] Failure to promptly and adequately refrigerate a body after death may also occasionally result in unexpected decomposition.[13] This may occur when bodies have been left at room temperatures in hospital beds for a number of hours (usually for family viewing), in vehicles at crash scenes, or in body storage rooms with inappropriately high temperatures. Accelerated decomposition was a feature in many victims of the South East Asian tsunami in 2004 due to a combination of high environmental temperatures, humidity, and inadequate refrigeration.

Another important factor is obesity, as subcutaneous and abdominal fat have insulating properties that slow the rate of cooling. For example, males cool more rapidly than females of identical weight due to the higher fat content in females.[2,8,14] Clothing and other coverings such as blankets also insulate a body from the environment and reduce conduction and convection.[8]

If the human body is regarded as a culture medium for bacteria, the same principles that apply in microbiology laboratories to enhance organism growth[15–17] will apply after death. An optimum temperature is required, with moisture to avoid desiccation.[3] The most favorable temperatures for putrefaction are between 21 °C and 38 °C with putrefactive activities impeded when the temperature is less than 10 °C[6,8]; certain putrefactive bacteria, such as clostridia, grow best at 37 °C. The actions of hydrolytic enzymes are enhanced by higher ambient temperatures, thus accelerating autolysis. Putrefaction is also enhanced by high environmental humidity.[5,6,16]

Elevated ambient temperatures also enhance the odor of putrefying bodies, thus attracting more insects, rodents, and carnivores. Breaches through the skin created by these creatures (e.g. bites) facilitate invasion by exogenous aerobic bacteria, which can accelerate putrefaction. Maggot infestations also increase the rate of putrefaction as insect larvae secrete a proteolytic enzyme that enhances tissue destruction.[7]

Decomposition is accelerated in deaths from infectious diseases due to the combination of increased antemortem bacterial load, preexistence of bacteria in the blood and organs, and the likelihood of an elevated temperature at the time of death.[2] Since bacteria spread most easily in fluids, putrefaction may be more rapid in edematous tissues arising from congestive heart failure, liver failure, or renal failure. Conversely, deaths from exsanguination and dehydration may delay putrefaction, as blood normally facilitates bacterial spread.[6] Considerable blood loss also leads to a deficiency of haemoglobin and other proteins from erythrocytes that are a significant source of energy for putrefactive bacteria.[2]

It was noted in this study that putrefaction was enhanced when significant hyperglycemia from diabetes mellitus was present (case 1). This has not been previously emphasized in the literature, however, it is known that putrefactive bacteria require organic carbon, which they can obtain from glucose via fermentation.[17] This requirement is reflected in artificial media and agars used to culture anaerobic bacteria that contain between 10 and 18 g of glucose per liter, and media that is used for the cultivation of *Clostridium* species, such as chopped meat-glucose and thioglycolate agar that contain 5.0 g and 5.5 g of glucose per liter, respectively.[18] Thus, estimation of time of death based on putrefactive changes in those dying with significant hyperglycemia may be even less accurate than usual.

Illicit and prescribed drugs such as the ring-derivate amphetamines (3,4-methylenedioxymethamphetamine or ‘ecstasy’), cocaine and drugs that may have an atropine-like effect such as benztropine may also cause significant antemortem hyperthermia.[9,11]

Table 1
Factors increasing the speed of decomposition of cadavers.

Exogenous				
	High ambient temperatures			
		Outdoors		
			Summer months + humidity	
			Fire	
			Vehicle	
				Carbon monoxide suicide
				Entrapment
		Indoors		
			Central heating	
			Bath/shower/sauna	
			Electric blanket/excess bedding	
			Fire	
	Failure (or delay) to refrigerate			
Endogenous				
	High initial core temperature			
		Fever		
		Drugs		
			Ring derivative amphetamines	
			Cocaine	
			Benztropine	
	Sepsis			
		Localized		
		Generalized		
	Obesity			
	Diabetes mellitus			

In conclusion, accelerated decomposition may confuse estimates of the time of death and/or throw doubt on the veracity of witness statements. Identification of any of the factors that enhance autolysis and putrefaction such as increased antemortem bacterial loading, elevated or maintained body temperature, obesity and increased nutrient availability should alert pathologists to the unreliability of using these findings to predict time of death. Information from the death scene, including ambient temperatures around the time of death may be extremely useful. With the increase in obesity and diabetes mellitus in Western communities,[14] and possibly in environmental temperatures generally, accelerated decomposition may become an increasing issue for pathologists, police and mortuary staff. Factors increasing the speed of decomposition of cadavers are summarized in Table 1.

Conflict of interest

The authors have no conflict of interests.

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